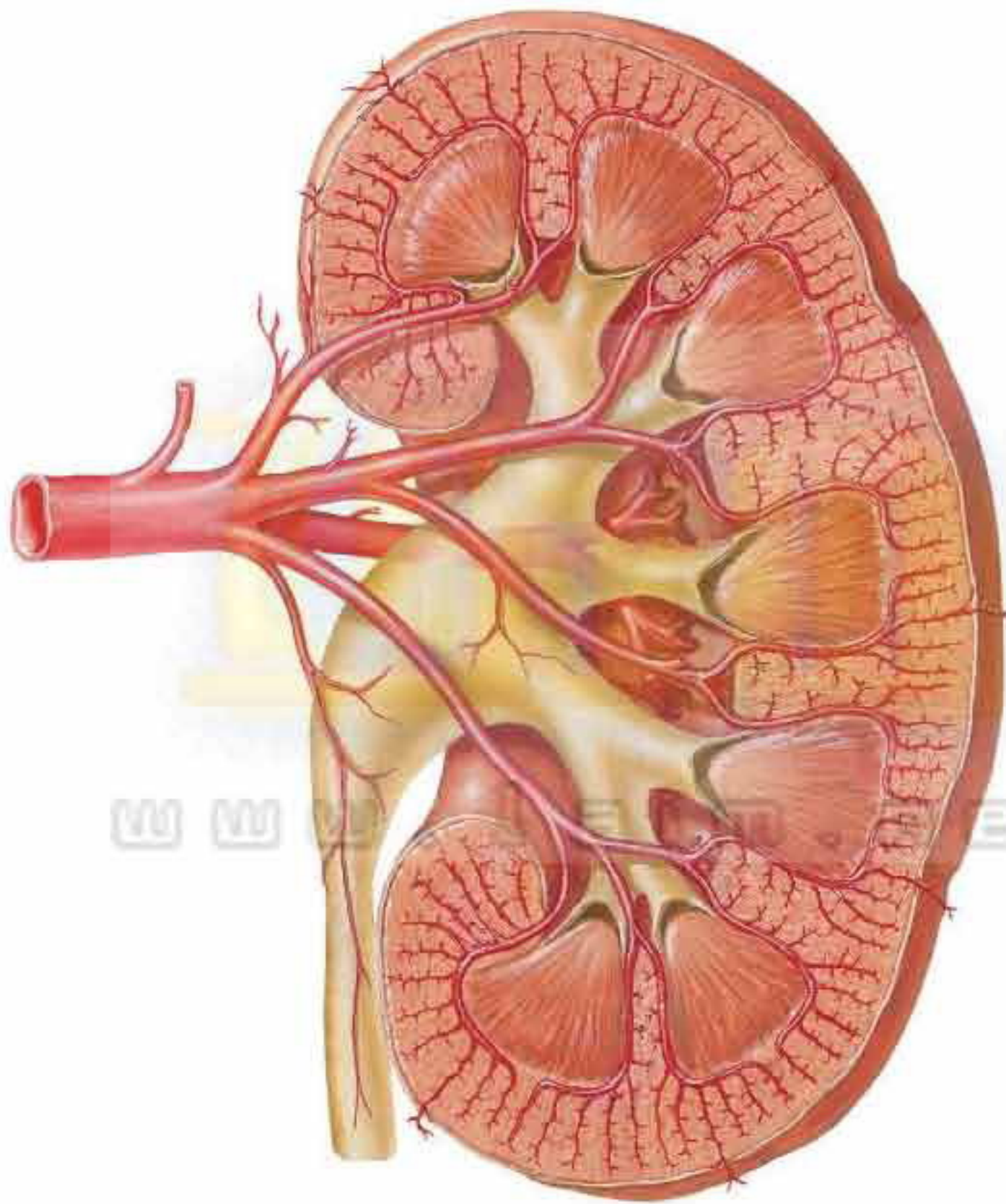


nephrology



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F. Netter M.D.
© IBN

Index:

- **CHRONIC RENAL FAILURE.**
- **ACUTE RENAL FAILURE.**
- **ACUTE TUBULAR NECROSIS.**
- **DIALYSIS & KIDNEY TRANSPLANT.**
- **GLOMERULO-NEPHRITIS.**
- **NEPHROTIC & NEPHRITIC S.**
- **TIN / UTI / TUBULAR DEFECTS.**
- **RAS – ADPKD – RENAL CARCINOMA.**
- **RENAL STONES.**

VISIT...

LANZAROTE
Caliente.COM

CHRONIC RENAL FAILURE = UREMIC \$

CHEST:

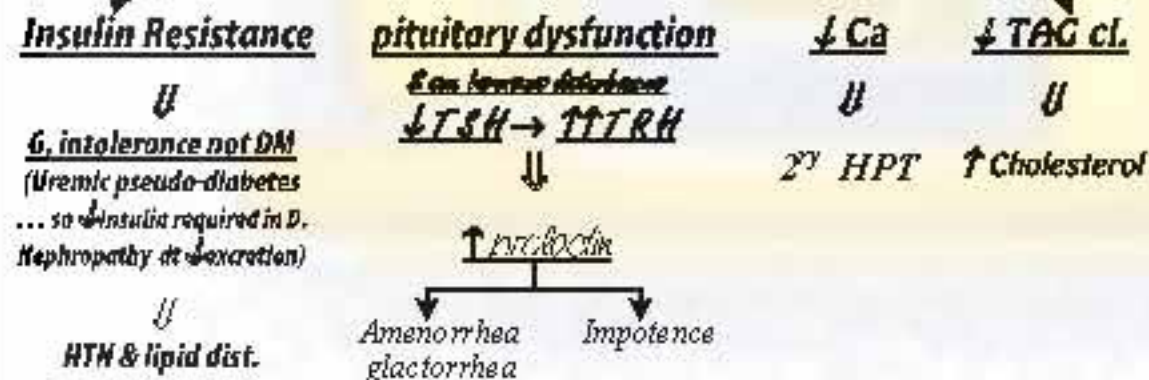
- 1) **pneumonia.** ($\downarrow$ IMMUNITY).
- 2) **Acidotic breathing.** (AIR HUNGER)
- 3) **NCPE.** (ARDS)

Gradual – slowly progressive
- Irreversible deterioration of KF $\Rightarrow$ ESRD.
(Requires Dialysis / Transplant)

CVS:

- 1) **pericarditis** dt * $\Rightarrow$ "painless" immediate dialysis.
- 2) **pericardial eff.** $\Rightarrow$ hgc e tamponade dt heparin used during HD.
- 3) **CARDIO-MYOPATHY** DT* $\Rightarrow$ HF
- 4) **ARRHYTHMIAS** $\Rightarrow$ dt El. (hyper-kalemia)
- 5) **HYPERTENSION** $\Rightarrow$ DT salt & H₂O retention.

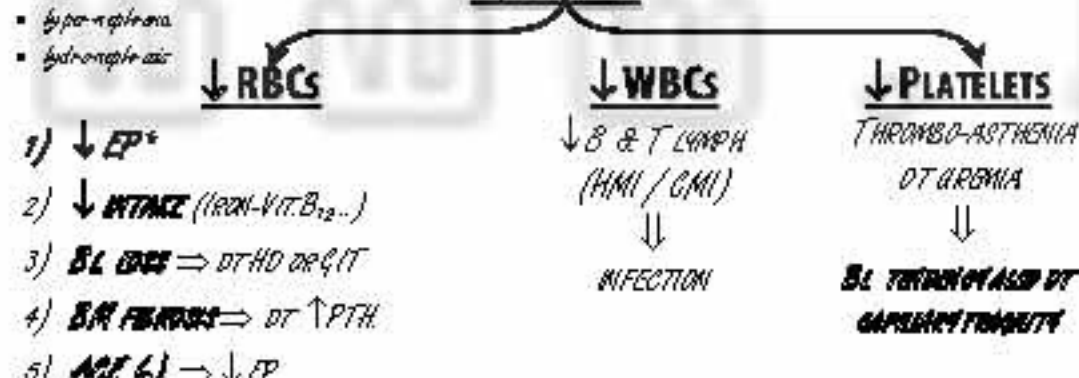
Endocrine



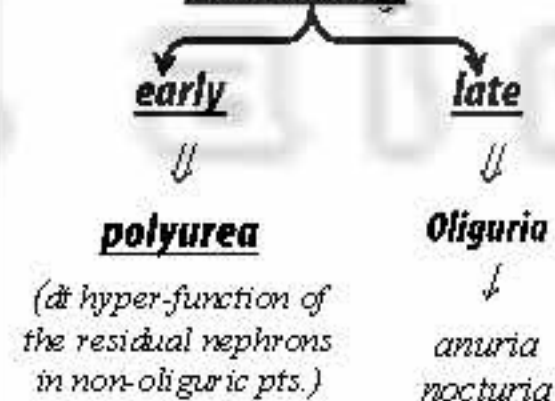
IF CRF + mild ANEMIA dt ↑EP

- poly-cyclic benzene
- hydro-aromatics
- hydro-aromatics

Blood



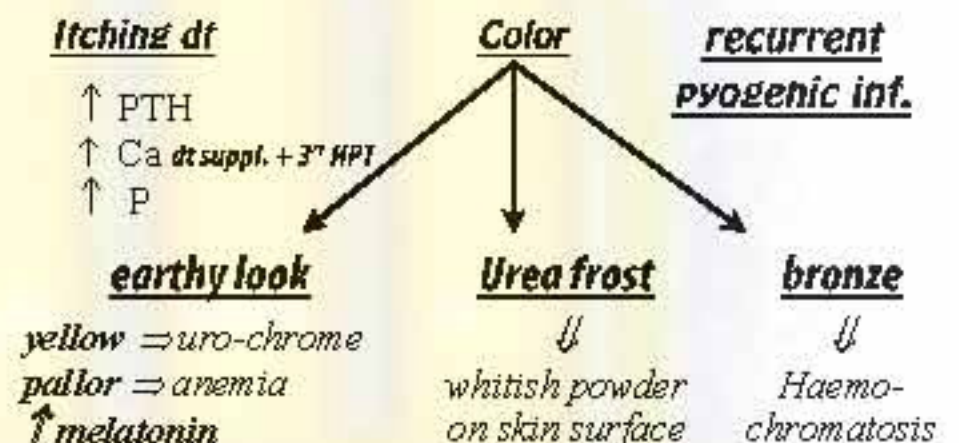
Urinary



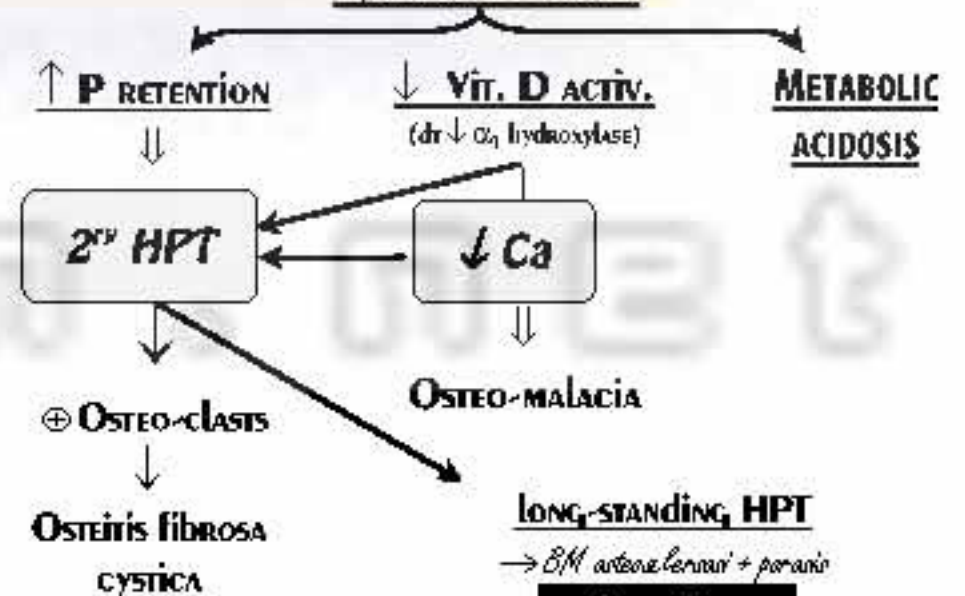
GIT:

- 1) **NH₃ odor**. (DT UREA SPLITTING IN SALIVA BY B. FLORA)
- 2) **NVD & Hi-cough** $\Rightarrow$ UREA $\oplus$ CTZ (persistent ~~not~~ responding to usual tx)
- 3) **Gastritis** $\Rightarrow$ DT* + $\uparrow$ GASTRIN.
- 4) **G I bleeding** $\Rightarrow$ $\downarrow$ BP $\rightarrow$ $\downarrow$ KID. PERFUSION
- 5) **Hepatitis (B-C)** $\Rightarrow$ DT BLOOD TRANSFUSION.

Skin



Bone = ROD



7- Neurological

Brain	Neuropathy	MUSCLES
UREA ENCEPHALOPATHY DIALYSIS STROKES <i>due to HTN & bl. tendency.</i> rapid ↓↓ urea AL toxicity BRAIN EDEMA DIALYSIS DEMENTIA Dis-Equilibrium \$ AS IN DM rapid ↓ IN BS.	MOTOR MS. WEAKNESS SENSORY Glove & Stock hypothesia AUTONOMIC α1 Rs. IMPOTENCE & ORTHOSTATIC hypoTN. MUSCARINIC D. & C. & GASTRO-PARESIS	- Uremic Myopathy - Uremic Myoclonus ⇒ Convulsions. UL CARPAL TUNNEL \$ dt Amyloidosis (B2 μ-globulin). LL RESTLESS LEGS \$ (irresistible need to move legs) BDZ

8-ACID BASE & ELECTROLYTES

- ☒ ↓ Ca
- ☒ ↑ K. ↑ P – ↑ AL. (bec. all ARE EXCRETED THROUGH kidney)
- ☒ Na NORMAL (EXCESS WATER INTAKE → Dilutional hypoNATERMIA)
- ☒ ↓ pH ⇒ METABOLIC ACIDOSIS DUE TO H⁺ RETENTION.

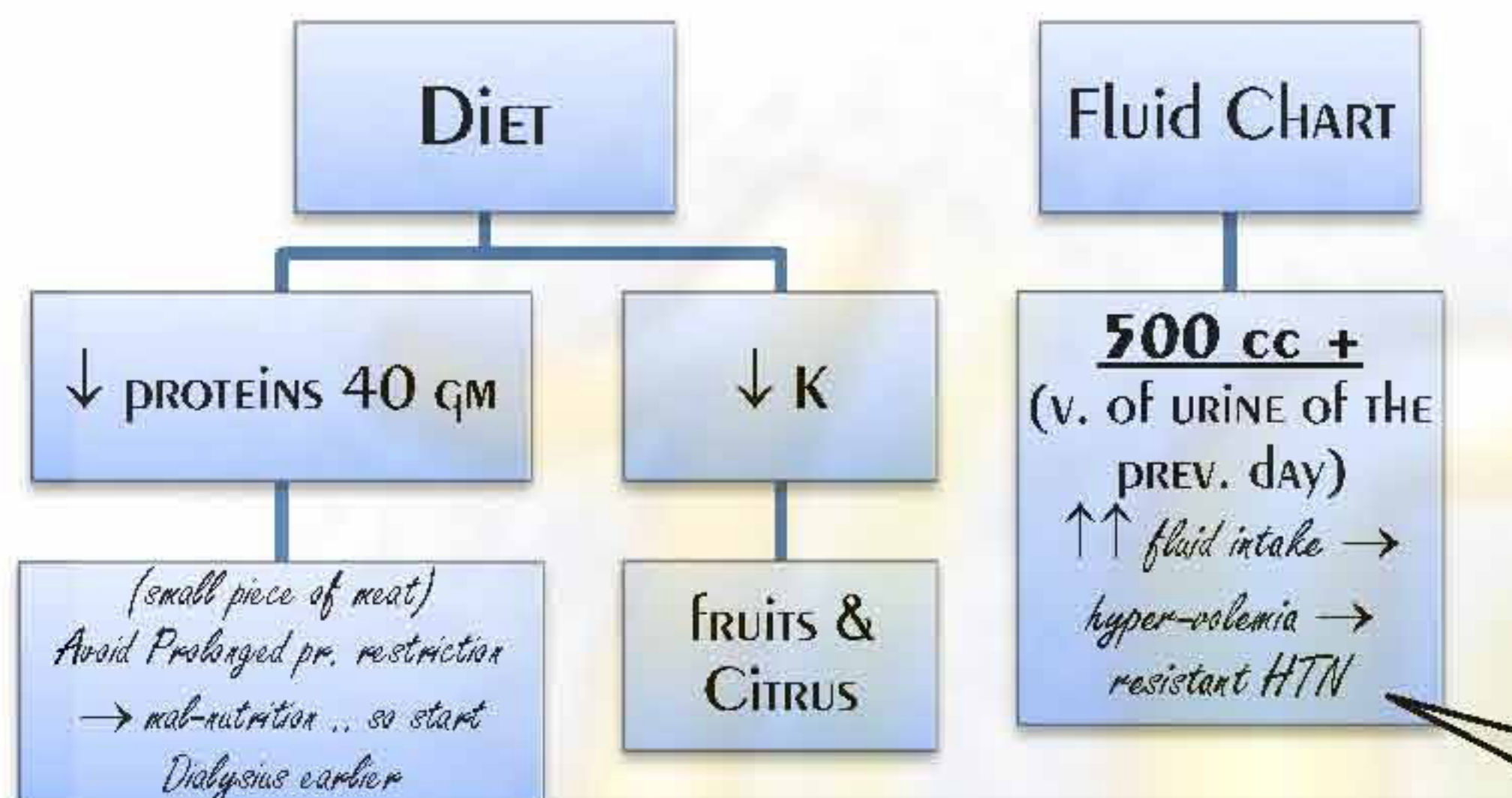
INVESTIGATIONS FOR CRF

To prove RF	To prove Chronicity	CAUSES (TO CONTROL ANY REVERSIBLE FACTORS)
• $\uparrow$ Urea & Cr. • $\downarrow$ PH, $\uparrow$ K	1) Blood: ($\downarrow$ Ca, $\uparrow$ P, $\downarrow$ Hb) <u>BUT MAY OCCUR E ARF.</u> <u>CARBAMYLATED hb</u> = NON-ENZYMATIC REACTION BET. UREA & hb $\Rightarrow$ CKD 2) URINE: • Broad casts. • Low <u>fixed</u> Sp. G (1010) = Iso-thenuria. 3) SONAR : (MOST IMPORTANT) <pre> graph TD SONAR[SONAR] --> SHRUNKEN[SHRUNKEN] SONAR --> ENLARGED[ENLARGED] SHRUNKEN --> SYMETRICAL[SYMETRICAL] SHRUNKEN --> ASSYMETRICAL[ASSYMETRICAL] SYMETRICAL --> ChGN[Ch. GN] ASSYMETRICAL --> ChPN[Ch. PN] ENLARGED --> List["1) Diabetic Nephropathy. 2) Amyloidosis. 3) hydro-nephrosis 4) Polycystic kidney"] </pre>	1) URINE: - pus cells $\rightarrow$ active bacterial infection. - RBCs casts $\rightarrow$ GN 2) X-Ray $\Rightarrow$ stone. 3) SONAR $\Rightarrow$ stones & hydro-nephrosis. 4) Blood: - Bl. Sugar $\Rightarrow$ Diabetic nephropathy. - Markers $\Rightarrow$ SLE - Eosinophilia $\Rightarrow$ Vasculitis. AGGRAVATING FACTORS IN UREMIA: (REVERSIBLE) - HYPOTENSION. $\Rightarrow$ $\downarrow$ KIDNEY PERFUSION. - NEPHRO-TOXIC DRUGS. (NSAID - AG) - UTI & OBSTRUCTION. - HYPERTENSIVE CRISIS. - SMOKING - HIGH PROTEIN DIET - CONTRAST NEPHROPATHY. (IVP / ANGIOGRAPHY / CT)

TREATMENT OF CRF

CONSERVATIVE = MEDICAL TTT	Dialysis
INDICATION: All 3 TOGETHER 1) Fair GC = minimal symptoms. 2) S. Cr. < 8 MG% (< 6 GM in Diabetics) 3) Blood UREA < 200 MG %.	INDICATION: 1 is ENOUGH 1) Bad GC. 2) S. Cr. > 8 AND > 6 in DM 3) BL. UREA > 200.

MEDICAL TREATMENT OF CRF



CVS:

- 1) HTN
 - BB / CCB. / ACE-I.
- 2) Peri-CARDITIS
 - INDICATION of Dialysis
 - NSAID (NEPHRO-TOXIC)
 - STEROIDS.
- 3) Hypolipidemics
- 4) HF & ARRHYTHMIA ⇒ (see C.V.S.).

Endocrine:

- ☆ Ca supply ⇒ $\text{Ca CO}_3 + \text{Vit D (1-}\alpha\text{)}$
- ☆ Anabolic STEROIDS for Bone D. + ↓ protein cat.
- ☆ ↑ prolactin ⇒ BROMO-CRIPTINE.

Blood "ANEMIA"

- IRON INFUSION.
- ERYTHROPOIETIN. If no response ⇒ Iron def. / Bleeding / Malignancy.
- Avoid Blood tr. to ↓ sensitization of HLA Aps. → Barrier in Renal Transplant + ↓ Risk of hepatitis B & C infection

RENO-PROTECTION TO Slow progress of RENAL D.?!?

- 1) Diet pr. RESTRICTION → G. SCLEROSIS.
- 2) D. NEPHROPATHY → ACE-I OR ARBS. (↓ IGP)
- 3) CONTROL BP / BS in DM.
- 4) Avoid NEPHRO-TOXIC DRUGS.
- 5) CORRECT ANY REVERSIBLE FACTOR.

HYPER-VOLEMIA:

- 1) ↑ V. pr. → CONGESTED Neck v.
 - 2) ↑ A. pr. → ↑ BP
 - 3) PE → DYSPNEA – ORTHOPNEA.
 - 4) RESISTANT HTN.
- ↓
- LASIX LARGE DOSE ± Ultra-filtration.
(80 mg IV / IM)

GIT:

- Vomiting & Hiccough ⇒ Domperidone.
If no Response ... start Dialysis immediately.
- Gastritis ⇒ H₂ blockers OR PPI.

Acid-BASE:

- ↓ K INTAKE + REMOVE FROM GIT
Sorbisterit (causes const. → LACTULOSE)
- Lasix & ↓ dose of ACE-I & ARBs
- If NO RESPONSE ⇒ Dialysis.

PRURITIS: CONTROL Ca/P balance + Anti-histaminics.

REVERSIBLE FACTORS:

- UTI ⇒ ABS
 - DM ⇒ control
 - Stone ⇒ surgery
 - HTN ⇒ control
- Monthly follow up (Cr., UREA, Na AND K, Hb ,Ca ,P)

Bone PHOSPHATE CHELATORS:

1) Ca CO_3 ACTS AS:

- a) Ca supplement
 - b) ↓ P absorption.
 - c) ttt. of Acidosis.
- S/E = constipationso give Ca acetate.

2) AL-OH MUST BE AVOIDED ⇒ ROD + DEMENTIA.

3) NON-Ca / NON-Al (SEVELAMER) 800 mg TDS before meals

	HAEMO-DIALYSIS	PERITONEAL Dialysis
AdvANTAGES (CONT. Medical TTT. of CRF)	1 <i>less invasive than peritoneal Dialysis.</i> 2 <i>Rapid action.</i>	1 <i>Natural membrane $\Rightarrow$ remove the middle molecule subst.</i> 2 <i>No heparin use.</i> 3 <i>No haemo dynamic changes.</i>
IndICATION	1) ARF & CRF. 2) $\uparrow$ K + $\uparrow$ Ca 3) DRUG TOXICITY. Esp. Alcohol. 4) ASCITES $\Rightarrow$ ULTRA-FILTRATION & REINFUSION. 5) PE $\Rightarrow$ ULTRA-FILTRATION.	1) BLEEDING TENDENCY. 2) DIABETIC NEPHROPATHY. 3) HYPOTENSION (bec it DOESN'T CAUSE hemodynamic disturbances)
Disadv.	1) INFECTION <i>(Hepatitis / HIV / IEC due to Central Jugular Catheter)</i> 2) BLEEDING. <i>(due to heparin + Thromboasthenia)</i> 3) BRAIN EDEMA. "Dys-equilibrium S" <i>(dt rapid $\downarrow$ Urea / Na / Glucose)</i> 4) Hypo-TENSION. 5) DIALYSIS DEMENTIA. <i>(dt Al toxicity)</i> 6) DIALYSIS Amyloidosis. & ACC. ATHERO-SCLEROSIS <i>(dt incompatible dialyzable membrane $\rightarrow$ bl. Reactions $\rightarrow$ Chronic inflam. state)</i> 7) AIR EMBOLISM.	1) INFECTION $\Rightarrow$ peritonitis. 2) DELAYED RESPONSE. <i>(time consuming)</i> 3) INJURY of viscous ORGAN.
CONTRA-INDICATION	❖ <i>Bleeding tendency.</i> ❖ <i>Hypotension.</i>	❖ <i>Urgent case. (Haemodialysis is preferred)</i> ❖ <i>Ascites.</i> ❖ <i>Peritoneal adhesions.</i> ❖ <i>Intra-Abdominal Sepsis.</i>

Kidney Transplantation

Pre-operative	Operative	Post - operative
1- A B O & H L A for Donor & Recipient. 2- Donor ⇒ Routine invest. (angio - scan - IVP) 3- Recipient ⇒ micitulating cystogram ⇒ to exclude reflux.	1- Nephrectomy ⇒ We try to shorten • Hot ischemia (time bet. clamping of renal vs & *) • Cold Ischemia (time bet. * & transplant). 2- Kid. perfusion ⇒ wash capillaries to ⊖ μ-thrombi. 3- Kid. transplant ⇒ anastomosis e iliac vs & uretero-vesical anastomosis.	(to ⊖ rejection) 1) Steroids. 2) Cyclosporine. 3) Mycophenolate.

Indications of Kidney Transplant = E S R D

- Ch. GN / PN.
- ADPKD
- Obstruction Uropathy.
- Amyloidosis.
- Chronic IN.
- Lupus Nephritis. (NOT A # FOR TRANSPLANT bec. tit. of lupus = tit. of Graft)

Common complication	Recurrence after transplantation
1) A. T. N. 2) Rejection 3) RV OR RA THROMBOSIS. 4) URINE LEAKAGE AT URETRO-VESICAL ANASTOMOSIS. 5) Complications from IMMUNE-SUPPRESSION. 6) RECURRENCE OF THE 1 ^{RY} RENAL DISEASE. eg. MEMBR. Prolif, FSGS.	1) MPGN. (Type II). 2) FSGN. 3) Good PASTEUR'S \$. 4) Type I Oxaluria. (liver problem dt ↑ production)

ACUTE RENAL FAILURE

sudden deterioration of kidney functions within hrs. or days - reversible with treatment of the cause

	PRE – RENAL = REVERSIBLE <i>"normal kidney with ↓ perfusion" ...functional disorder only</i>	RENAL <i>"Intrinsic parenchymal renal disease"</i>	POST- RENAL <i>"Obstruction → interference"never fluids</i>
<u>Causes</u>	1) hypo volemia (↓ BP + ↓ VP) • blood → hge. • plasma → burns. • fluids → severe diarrhea - pancreatitis. 2) Shock + Normal volume (↓BP + ↑ VP) • Cardiogenic shock (extensive MI) • Massive pulm. Embolism. (from DVT) 3) SEROUS SAC: pancreatitis - hepato-renal S. 4) DRUGS= (NSAID ... ↓ PG → RENAL ISCHEMIA) & (ACE-I → ↓ IGP → ↓ GFR)	1) ATN. "neglected pre-RF" → casts 2) ACUTE IN → Eosinophilia -uria. 3) ACUTE SEVERE PN e papillary necrosis → in DM. 4) RPGN → RBCs casts. 5) Malignant HTN → ↑ IGP	1) Bi-lat. URETERIC obst. 2) Uni-lat. URETERIC obst.
<u>CL./P</u>	1) OF THE CAUSE. 2) NO MANIFESTATIONS OF UREMIA.	1) OF ARF. (Oliguria + HTN) + NV .. Acidotic br. + Uremic Enceph. + hyper-volemia e PE) 2) OF THE CAUSE.	ACUTE ONSET of • Renal colic – ANURIA • DETERIORATION
<u>INVEST.</u>	1) ↑↑↑ UREA > Cr. dt partial re-absorption of urea by healthy tubules. 2) ↑ BUN / Cr. Ratio > 20:1 dt.. see above 3) ↓ Na in URINE → dt ↑↑ REAB. TO COMPENSATE THE ↓ BP. 4) UA → NO CELLS NO CASTS. 5) SONAR → NORMAL	1) ↑ UREA NOT HIGH dt damage of tubules → can't reabsorb Urea → Urea not high → BUN not high. 2) BUN NOT SO HIGH. 3) CASTS • GRANULAR → ATN • RBCs → Acute GN. 4) ACUTE IN → EOSINOPHILIA -URIA.. 5) VASCULITIS → +VE ANCA & ↑ ESR 6) SONAR → NORMAL SIZE & ↓ ECHOGENECITY.	pt. E. Oliguria & ANURIA for 2days INSERT URINE CATHETER URIN Output NO URINE OUTPUT RETENTION ARF مسالك بولية "PROSTATE" SONAR -VE +VE DIURESIS post-RF URINE OUTPUT NO URINE OUTPUT PRE-RF RENAL "ATN"
<u>III.</u>	1) OF THE CAUSE: • EXT. MI → Dopamine • Pulm. Embolism → Thrombolytic Th. 2) CVP → Adjust fluid intake 3) Dopamine (small dose) → VD of afferent a.	1) GENERAL: أكل و شرب و حرف الـ H و لو ترنقت اعسل 2) OF THE CAUSE • RPGN → Pulse Steroid th. • ACUTE TIN → Stop Nephro-toxic drugs. • ACUTE PN → ABs	INTERFERENCE. (ureteric catheter / stents / nephrostomy)

ACUTE TUBULAR NECROSIS = ATN

	Oliguric phase (2-4 wks)	Polyuric phase (3-4 days)	Post Diuretic phase
Causes:	Ischemic ATN "Diffuse affection" dt NEGLECTED pre-RF Toxic ATN "mainly PCT" Endog. Exog. 1) Hb. (VIT = G6PD + AIIIA + CRUSH \$) 2) Mb. (Rhabdomyolysis) 3) Bilirubin Neph. (Obst. J) 1) CONTRAST 2) DRUGS: AG. 3) G-VF SPECIFIC-MIA.	1) Relief of Tubular Obst. 2) REGENERATION of kid. Tubules but still UNABLE TO CONC. URINE. 3) UREA RETENTION. (Osmotic diuresis)	
CL/P = MANIFESTATION OF THE CAUSE + ARF			
Urine vol. GIT CVS METABOLIC	- OligUREA / ANURIA. - N V. - HYPERTENSION. - Hyper-vol. ⇒ PE. ↑K ⇒ WEAKNESS – ARRHYTHMIA. - Acidosis ⇒ ACIDOTIC BREATHING Differs from CRF in: - No ROD. - No ENDOCRINAL. - No ANEMIA.	1) Clinical improvement. 2) ↓ Na, ↓ K due to diuresis. 3) URINE output → 3-5 L / d	1) Clinical improvement. 2) KFT → NORMAL. 3) URINE out put → NORMAL.
INVEST. (no biopsy needed) 1) Blood ⇒ ↑Urea & Cr. (↓pH - ↑K) ↓Ca & ↑P. ↓Hb & sometimes normal. 2) URINE ⇒ Granular muddy Casts (ATN + Chronic GN dt dilatation of healthy nephrons) 3) SONAR ⇒ almost normal kid.			
NEGLECTED CASES of ARF ATN Reversible CORTICAL NECROSIS IRREVERSIBLE dt CRF			
TREATMENT: (AT REQ المريض حتي الـ)			
نغسل لو اتزنقنا 1) ↑K / Acidosis. 2) Hyper-volemia. 3) Pericarditis.	حرف الـ ↑H⁺ ⇒ Na HCO₃ - Hyper-k ⇒ Glucose + Insulin - Hyper-vol. & HTN. - Hyper-ph ⇒ Ca + AL OH	شرب ↓ Fluid chart 500 cc + v. of urine in the prev. day.	أكل ↓ ↓PROTEIN & ↓K
		1) Fluid 2) Na & K supplements.	Indications of HD in ARF: - HYPER-KALEMIA. S. ACIDOSIS. - HYPER-VOL. → PEE. - PERI-CARDITIS. (EMERGENCY)

DD

- **Appendicitis.**
- **Peri-Nephric Abscess.** no urinary symp. or finding only loin pain & tenderness.

Acute PyeloNephritis

"infection in interstitial t of renal parenchyma that may reach to renal pelvis & mucosa."

PDF

ppt. factors

- 1) **SEX:** ♀ > ♂ - short urethra + no bactericidal prostatic fluid.
- 2) **Obstruction**
 - young → stone, B.
 - old → prostate++
- 3) **PREGNANCY**
 - Hormones → relaxation of ureters → stasis.
 - pressure of uterus → stasis.
- 4) **NEUROGENIC bladder** → eg paraplegia
- 5) **CATHETER** → Nosocomial infection.
- 6) **VUR: esp. in Children**
 - Recurrent UTI → VUR.
 - Reflux nephropathy (FSGN) → Nephrotic S.
- 7) **RENAL DISEASES** oligurea → ↓washout of urine → infection.
- 8) **SYSTEMIC DISEASE** → DM.

CA

E. Coli.
Klebsiella.
PROTEUS.
Staph. -
ENTEROCOCCUS

ROUT of INFECTION

- 1) Asc. from URETHRA & UB.
- 2) HEMATOGENOUS.
- 3) Lymphatic.

RECURRENT UTI
SINCE CHILDHOOD

NO RESPONSE TO ABS

EDEMA ... puffiness

UA: HEAVY-PROTEINURIA

VUR

RECURRENT UTI

FSGN

DD FEVER + RIGORS

- ACUTE PN.
- HEMOLYTIC CRISIS.
- Asc. CHOLANGITIS.
- MALARIA.
- AMOEBIC LIVER.
- PYOGENIC ABSCESS

Cl/P. of Pyelo-Nephritis

Classic

Child

Pregnancy

DM

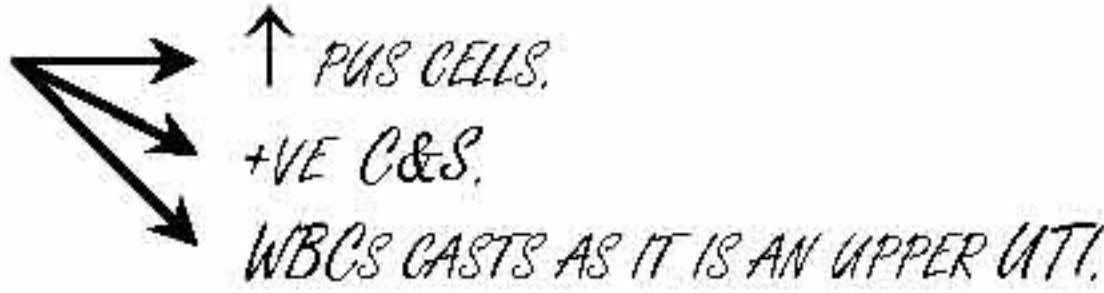
- 1) FAHM - Rigors
- 2) Loin pain & tenderness.
- 3) Dysuria, frequency & turbid urine.

FEVER & OUT LOCALIZATION
→ reflex vomiting
→ misdiag. as GE.

- 1) **ASymptomatic.**
- 2) **BACTERIURIA** > 100,000 ml → 40 % PN.

Acute Necrotizing
papillitis.
→ ARF.

INVESTIGATIONS FOR ACUTE PN:

- 1) URINE 
 - ↑ PUS CELLS.
 - +VE C&S.
 - WBC'S CASTS AS IT IS AN UPPER UTI.
- 2) Blood ⇒ ↑ ESR / CRP / TLC "DT INFECTION IN PARENCHYMA"
- 3) INVEST. THE CAUSE ESP. IN ♂
 - **PLAIN UT** → stone.
 - **IVP** → stone, stricture.
 - **MICTURATING CYSTOGRAM** (for reflux).
 - **SONAR.**

TREATMENT

- 1) ABS. (CO-TRIMOXAZOLE = **SUTRIM OR AMOXACILLIN**)
- 2) THEN AB ACCORDING TO C&S FOR 7-10 days.
- 3) REPEAT CULTURE DURING TIT & AFTER 7 & 21 days FROM TIT
 - a) Same org. within 7 d → relapse.
 - b) Same or other org. after 2 wks & -ve Bacteriuria → re-infection.

(IF NO APPARENT CAUSE OR RESISTANT DRG. → AB FOR 2-6 WKS ACCORDING TO C&S)
- 4) ALTERATION OF PH:
 - a) Alkaline urine (Na HCO_3) ⇒ potentiate **Sulpha + AG.**
 - b) Acidic urine (vit C) ⇒ potentiate **TETRACYCLINE.**
- 5) FLUID INTAKE:
 - ↑ urine output → ↑ washout of urine.
 - Dilute the urinary conc. of Ab → harmful ?!

DRUGS USED IN UTI:

• CO-TRIMOXAZOLE	2 tab / 12 hrs	
• Ampicillin	500 mg / 6 hrs.	
• AMOXACILLIN	500 mg / 6 hrs. (better than Ampicillin)	
• NITROFURANTOIN	100 mg / 8 hrs orally.	} # in RENAL D. → P. NEUROPATHY
• Nalidixic acid	500 mg / 6 hrs.	
• GARAMYCIN	3 mg / kg / d in divided dose.	
• NEPHROTOXIC	Amp 80 mg / hrs.	
• 3rd G CS	Cefotaxime 2 gm / d IM & IV (minimal nephrotoxicity)	
• Ofloxacin	400 mg / 12 hrs.	

prophylaxis in ♀ with RECURRENT UTI

- ✓ Fluids > 2L/D
- ✓ Regular emptying of UB / 3 hrs.
- ✓ Double micturation if reflux present.
- ✓ Empty UB before & after intercourse.

Chronic PyeloNephritis

"DUE TO CHRONIC STAGNATION = VUR OR OBSTRUCTIVE "STONE / STRICTURE"

CL./P:

- 1) HISTORY OF RECURRENT UTI.
- 2) POLYURIA (NO RESPONSE TO ADH) = NEPHROGENIC DI
or INFECTION IN INTERSTIAL.
- 3) CRF.

FARMER WITH B, RECURRENT UTI

↓
VOMITING – ANEMIA

HTN – ASICTES.

↓
CRF.

INVESTIGATIONS:

- 1) URINE → +ve C&S, pus (few) + WBCs casts +
(No RBC casts as no GN)
- 2) OF THE CAUSE:
 - a) *SMAV* → SHRUNKEN KIDNEYS "ASYMMETRICAL"
 - b) *NP* → CLUBBED CALYCES "BETTER TO BE AVOIDED ESP. IN RENAL D."
 - c) *MICTURATING CYSTOGRAM* (FOR REFLUX).

TREATMENT:

- 1) ABS ACC. TO C&S FOR 7-10 DAYS
- 2) LONG TERM SUPPRESSIVE THERAPY FOR 3-6 MS. (v. LOW DOSE & FOLLOW BY C&S)

E.g.

AMOXICILLIN 250 MG / D.

NITROFURANTOIN 50 MG / D.

TRIMETHOPRIM 100 MG / D.

}

URINARY ANTI-SEPTIC DOSE
- 3) INTRA-VAGINAL ESTROGEN → POSTMENOPAUSAL WOMEN.
- 4) REFLUX IN CHILDREN → URETERAL RE-IMPLANTATION IN BLADDER TO FORM A COMPENSATED VALVE.

NB: Calculate the dose of AG acc. To s. Creatinine.

Nephrotic \$

PROTEINS LOST IN URINE: LMWP

- A) ALBUMIN
- b) AT-III - PROTEIN C,S.
- c) Ig
- d) Thyroglobulin.
- e) TRANSFERRIN → IRON def. AN.

Injury to the glomerular barrier

heavy proteinuria
($> 3.5 \text{ gm} / 24 \text{ hrs.} / 1.73 \text{ m}^2$)

hypo-proteinemia
"selective to LMWP"

↓ Oncotic pr.

loss of fluid in ISF

edema

"Gradual / marked / Generalized"

starts as puffiness

"max. at the morning"

SEROUS TRANSUDATION

- pleural eff.
- pericardial eff.
- Ascites.

↓ IV Volume

⊕ RAS

↑ Aldosterone
↑ ADH

± hyper-lipidemia
(dt ⊕ of hepatocytes to synthesize HMWP)

hyaline or fatty casts

No ↓ GFR
"except late"

No Oliguria

No Azotemia
No HTN Also as BP is balanced bet.

↓ IV Volume
↓ BP

⊕ RAS
↑ BP

Nephritic \$ (Acute GN)

Immune complex deposition at the glomeruli

mild injury to the glomerular barrier

hematuria

RBCs Casts
Dysmorphic

Smoky URINE = DARK URINE
"dt passage of RBCs through Glomeruli then Tubule"

mild proteinuria

Edema¹

mainly endothelial & epith. proliferation

marked ↓↓↓ GFR

⊕ RAS

↑ Aldosterone
↑ ADH

SALT & WATER RETENTION

Edema²

Oliguria

Azotemia

Water Retention

HTN & Edema³

MILD INFLAM. → No Active Urinary sediments.

MARKED INFLAM. → Active Urinary sediments. (RBCs – WBCs)

ccc. by Gradual onset

- heavy-proteinuria. $> 3.5 \text{ gm} / 24 \text{ hrs} / 1.73 \text{ m}^2$
- hypo-proteinemia.
- edema (massive & generalized)
- ± hyper-cholesterolemia (dt ↑ lipo-protein synthesis by hepatocytes)

Nephrotic \$

+ Active Urinary Sediments:
(dt SEVER INFLAM. in Glomeruli)

Mixed GN

- A) MPGN.
- b) Mesangio-prolif.

ccc. by sudden onset (days)

- haematuria RBC Casts + Dysmorphic.
- oligurea.
- edema (mild & localized)
- hyper-tension & Azotemia

Nephrotic \$

Nephritic \$ (Crescentic GN)

CAUSES	1 st GN	2 nd GN	1) <u>post. Infectious:</u>
	MINIMAL LESION.	Diabetic / Amyloidosis.	a) Sept 1-3 wks after sore-throat.
	MEMBRANOUS GN	SLE – RA.	b) Staph- pneumo - viral.
	FSGS	Infections: B – MALARIA.	2) SLE. IEC.
	MPGN	Drugs: PENICILLAMINE – CAPTOPRIL	3) VASCULITIS. SHUNT NEPHRITIS.
	MESANGIO-PROL.	Malignancy: LYMPHOMA.	4) HSP. CRYO-GLOBULINEMIA.

INVESTIGATIONS

1. Blood	• $\downarrow\downarrow$ S. ALBUMIN.	• MARKERS (SLE, ASOT, ANCA)
2. Urine	• proteinuria > 3.5 gm / day • casts → hyaline or fatty . (dt hyper-lipidemia)	• proteinuria < 3 gm, $\pm$ > 3 gm. • casts → RBCs + WBCs . (dt inflammatory aetiology)
3. Renal F.	• UREA - CR - BUN $\Rightarrow$ NORMAL. (dt mild $\downarrow$ GFR except late $\uparrow\uparrow$)	• $\uparrow\uparrow$ UREA. CR. (dt MARKED $\downarrow$ GFR)
4. Biopsy		in RPGN suggesting crescentic GN

TREATMENT

CONSERVATIVE - edema $\rightarrow$ DIURETICS. (Lasix- Aldactone) - hyper-cholest. $\rightarrow$ hypo-lipidimics. (Statins) - proteinuria $\rightarrow$ ACE-I (early in DM) $\rightarrow$ CONTROL BP $\pm$ SUGAR $\pm$ DIET. (PR. RESTRICTION ESP IN DM)	IMMUNO-SUPPRESSIVE (STEROID + OTHERS) Fluid chart: dt “oliguria” - control BP. - salt restriction - protein restriction only in severe uraemia. - HTN / EDEMA $\rightarrow$ Diuretics & BBs. - of the CAUSE. - of Complications
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COMPLICATIONS

Nephrotic \$ + Dyspnea $\Rightarrow$ 4 Ps Pleural eff. $\Rightarrow$ Snov dullness. Pericardial eff. $\Rightarrow$ Tamponade ... heparatory fling of neck v. Pulm. Emb. $\Rightarrow$ Chest pain + hemoptis. ($\downarrow$ ATIII / Protein C & S $\Rightarrow$ DVT) PNEUMONIA $\Rightarrow$ FAHM + Chest pain + Dyspnea. (dt LOSS OF Ig IN URINE + STEROID + CYTO-TOXIC DRUGS $\rightarrow$ IMMUNE-DEF.)	hypertensive Encephalopathy. “fits” HF $\rightarrow$ $\downarrow$ BP PE dt “oliguria” $\rightarrow$ HYPER-VOLEMIA $\rightarrow$ PE $\rightarrow$ DYSOPNEA & ORTHOPNEA RPGN.
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PROGNOSIS

DD of Generalized edema CARDIAC / hepatic / RENAL.	post infectious GN - Children $\Rightarrow$ 90 % RECOVER. - Adult $\Rightarrow$ 50 % RECOVER. - SOME CASES $\Rightarrow$ RPGN / CHRONIC GN.
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	Nephrotic GN					Nephritic GN	
	MINIMAL LESION	MEMBRANOUS	Focal Segmental	RENAL Amyloidosis	DIABETIC	Diffuse proliferative	RPGN
Age	children not true GN at absence of inflam. 20% Adult	Adults (MC cause) if old age $\Rightarrow$ malignancy.	children (2 nd MC cause) Adults also.	not GN but Glomerulopathy	Not GN but part of D. Triopathy duration & control dependant (LASER – GLOVE & STOCK.)	(ACUTE GN & M/C CAUSE OF Nephritic \$) 1) post-Infectious: (strept & non- strept 2 wks after inf) 2) SLE 3) HSP: common in children • Arthritis • Abd pain • hematuria • purpuric eruption on buttocks & legs 4) Shunt Nephritis. (in hydro-cephalous)	Nephritic \$ + ARF (Rapid $\uparrow\uparrow$ s. Cr. = EMERGENCY) Infectious: 1) post streptococcal. 2) infective endocarditis. MULTI-SYSTEM DISEASES: 1) SLE 2) HSP. 3) Wegner's Granuloma. 4) Good pasture \$ DRUGS: 1) Hydralazine- Rifampicine 2) penicillamine 1 st Glomerular D. : $\pm$ ANCA
ccc. by	ccc. by • STEROID SENSITIVE. • SELECTIVE PROTEINURIA • SPONT. REMISSION. • NO CRF.	ccc. by • heavy PROTEINURIA ... (AT-III, Protein C, S) • $\uparrow\uparrow$ RV Thrombosis (loin pain... Duplex scan + sonar) • CRF IN 5-10 YRS.	ccc. by • STEROID RESISTANT • CRF IN 10 YEARS.	ccc. by • NEPHROTIC \$ $\rightarrow$ DETER. OF KIDNEY F. $\rightarrow$ CRF	ccc. by MC CAUSE OF ESRD • DM + Nephrotic\$ + HTN. • Type I > II at long history • persist. ALBUMINURIA STAGES of D. Nephropathy • hyper-filtration. ($\uparrow$ GFR > 150) (related to glycemic control) • μ -albuminuria. (30-300) • Gross proteinuria. (>300) • Renal impairment. • ESRD.		
Causes	1 st $\Rightarrow$ Idiopathic. 2 nd $\Rightarrow$ NSAID - lymphoma	1 st $\Rightarrow$ Idiopathic. 2 nd $\Rightarrow$ • SLE. • Captopril (large dose for long duration) • Gold – penicillamine. • HBV, malaria, Malignancy (old age)	1 st $\Rightarrow$ Idiopathic. 2 nd $\Rightarrow$ HIV, Heroin ESRD < 1yr. VUR, B, SCA.	1 st $\Rightarrow$ MULTIPLE MYELOMA. 2 nd $\Rightarrow$ INFECTIONS + CH. INFLAM. • RA (M/C CAUSE) • supp. lung (Chronic) • FMF: peritonitis (recurrent fever + Abd. pain + rebound tenderness)			
➤ MICRO							
LM	Normal (Nil \$)	Thick BM	Normal	Stain by Congo red $\rightarrow$ pink.	Thick BM	$\uparrow$ cells (epith + inflamm.)	CRESENTIC GN $\uparrow$ Cells (epith... + inflam.) Good prognosis & START PULSE Steroid IMM. $\uparrow$ fibrous T. Bad prognosis ESRD
EM	• fusion of foot processes. or Effacement.	• Thick BM at deposition of IC. • Sub-epithelial spikes.	• Focal $\Rightarrow$ some glomeruli affected. • Segmental $\Rightarrow$ part of the glomerus. • Sclerosis $\Rightarrow$ hyaline material (Thick wall / NARROW LUMEN.... HTN)	polarized MICRO $\rightarrow$ green Bi-refringence SONAR $\rightarrow$ ENLARGED kidney although CRF	hyaline arterio-sclerosis of aff. & efferent arterioles	Sub-epithelial humps (due to IC deposits) (If sub-epith $\rightarrow$ infectious cause)	
IF	*	IgG, C ₃	IgM, C ₃	*	* so renal biopsy isn't imp.	IgG, C ₃	
➤ TREATMENT OF ANY NEPHROTIC \$							
CONSERVATIVE				IMMUNO-SUPPRESSIVE (Steroid + Others)			
1) edema $\rightarrow$ Diuretics. 2) hyper-cholesterimia $\rightarrow$ hypo-lipidimics. 3) proteinuria $\rightarrow$ ACE-I. $\rightarrow$ (in early stages in DM to $\downarrow$ Glomerulo-sclerosis) 4) CONTROL BP $\pm$ SUGAR $\pm$ DIET. $\rightarrow$ (pr. restriction esp in DM at hyper-filtration)				RENAL D. + HAEMOPTYSIS 1) Wegner's Granuloma 2) Good Pasture \$. 3) TB.			
Good Response TO STEROIDS: 60 MG 4 wks $\rightarrow$ till proteinuria disappear. 2 wks $\rightarrow$ after urine become free 4 wks $\rightarrow$ gradual withdrawal. If NO Response OR Relapse...Add a) Cyclo-sporine. "BETTER" b) Cyclo-phosphamide.	PROTEINURIA + RENAL SCAN MILD TO MODERATE + N. GFR $\downarrow$ أكلأشيه وبس MODERATE + $\downarrow$ GFR $\downarrow$ STEROID only isn't effective so Add... a) Chlorambucil. b) Cyclosporine.	No Response TO Steroid th. 1st FSGN $\downarrow$ أكلأشيه + 1st STEROIDS only 2nd FSGN $\downarrow$ أكلأشيه + ttt of cause 2nd ... + Cyclosporine 3rd ... + Mycophenolate	أكلأشيه + the cause 1st $\downarrow$ STERIODS & Melphalan. FMF $\downarrow$ Colchicine $\downarrow$ TTT. of FMF $\downarrow$ PROGRESSION.	أكلأشيه (Esp. ACE-I)	of the cause: • infection $\rightarrow$ ABS • SLE $\rightarrow$ Steroids of Nephritic \$: • fluid chart • anti hypertensives • Diuretics Prognosis: • Good in children. • v. bad in elderly.	PULSE STEROID IMMEDIATELY IF SUSPECTED + القاعدة كاملة + PLASMA PH. GOOD PASTURE \$ AUTOIMMUNE $\rightarrow$ ANTI-GBM AB SAME AS RPGN + HAEMOPTYSIS (dr similarity bet. GBM & Alveolar BM) • LM: crescents "c below" • EM: epith. + inflam. cells. • IF: no IC + linear deposits of IgC TTL AS RPGN	

1611

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MIXED GN

**Nephrotic Range proteinuria +
Active Urinary sediments.**

MEMBRANO-PROLIF. GN

MESENGIO-PROLIF. GN

Causes

1st Idiopathic MPGN.

2nd to Infectious D.:

- a) **HCV** e Cryoglobineamia,
- b) **B - Infective Endocarditis**. (RPGN / MPGN)

• **SLE**, Neoplasm (leukemia – lymphoma)

1st Idiopathic MPGN.

2nd to SLE

Bilharziasis

IgA nephropathy (Burger's disease)

Kidney & SORE-THROAT:

- 1) **Post-streptococcal**.
- 2) **IgA Nephropathy**.
- 3) **ABS Abuse** → **Acute HS TIN**
→ **HS eosinophiliauria.**

CL./P

- 1) **Nephrotic \$ only** → **HEAVY PROTEINURIA RANGE.**
- 2) **Nephritic \$ only** → **ACTIVE URINE SEDIMENTS.**
- 3) **MIXED.**

*recurrent painless haematuria
in child & young adult with URTI*

IgA Nephropathy
(Burger's D.
is part of HSP)

+ purpura on buttocks

HSP

recurrent hematuria

painless

painful

- 1) **IgA Nephropath.**
- 2) **HSP.**
- 3) **Alprt's \$.**
- 4) **CM disease.**
"benign familial
hematuria"

Vasculitis.
ADPKD

RENAL STONE
ADPKD
B.

MICRO

LM

Thick BM + prolif. of mesangial cells.

Mesangial proliferation.

EM

Type I - Sub-endothelial depositis.

Mesangial proliferation.

IF

IgG – C₃ (↓C₃ + Normal C₄ as SLE.)

IgA – C₃ in mesangium.

TREATMENT

أكلاشة + No response to drugs

- 1) **Plasma -ph.** "of choice"
- 2) **Steroids + Interferon ?!** (if HCV)

DD of ↓ Complement in GN

- **MPGN.**
- **SLE.**
- **post-infectious.**

- 1) **Steroids** (proteinuria > 3 mg/d)
- 2) **Fish oil.** (Omega 3)
- 3) **Long term Ab.**

NB: **Recurrence of GN in transplanted kidney** ⇒ **FSGN / MPGN. (Type II)**

Tubulo Interstitial Nephritis

Inflammation of the renal interstitial tissue

early



Tubular damage but
(Glomeruli spared)

late



↑↑ Interstitial Fibrosis
(+ Hyalinized Glomeruli)

NB: pyelonephritis = interstitial nephritis caused by infection.

	Acute Interstitial Nephritis (Drug Induced HS Nephritis)	Chronic Interstitial Nephritis
<u>CAUSES</u>	a) ABS → Penicillins, CS, Sulpha – Rifampicin. b) Furosemide, Allopurinol. c) NSAID. d) Acute PN.	1) NSAID abuse. (♀ & Chronic headache) 2) SCA. 3) Lead. Irradiation. 4) Gouty Nephropathy. 5) Chronic PN.
<u>DIAGNOSIS</u>	1) ARF. Oliguric → Acute GN. Non-Oliguric → Acute IN. 2) Allergy → (fever - rash), arthralgia. 3) hyper-sensitivity → Eosinophilia & Eosinophiluria. 4) PCT defects → Glucosuria, Aminoaciduria, Phosphaturia. 5) DCT defects → Hyperkalemia - RTA. 6) Proteinuria → < 1 gm/dt tubular damage.	1) Of the cause. 2) of CRF. 3) polyuria due to defects in urine concentration. 4) Proteinuria usually < 1 gm. (قليل dt tubular damage) 5) salt wasting may occur.
<u>TTT.</u>	1) Stop drugs ± short term steroid? 2) Dialysis for A.R.F.	1) of cause. 2) Treated as C.R.F.

	Upper UTI (Pyelo-Nephritis = parenchymal D.)	Lower UTI (Prostatitis, Cystitis, Urethritis)
C/P	- Loin pain. - Toxaemia. (fever + Rigors) ± Dysuria.	- No Toxemia. - Dysuria, frequency
BL. Picture	↑ TLC / ESR / CRP	x
Urine	↑ pus cells. + ve Culture. - WBC cast + ve - Organism coated with Ab.	✓ ✓ x x Urine Analysis ↓ Pyuria ↓ C&S ↓ +ve ↓ Infection -ve ↓ GN TB Under ABS. Renal Stones.
TTT.	Long term	Short term

Tubular Defects of the Kidney

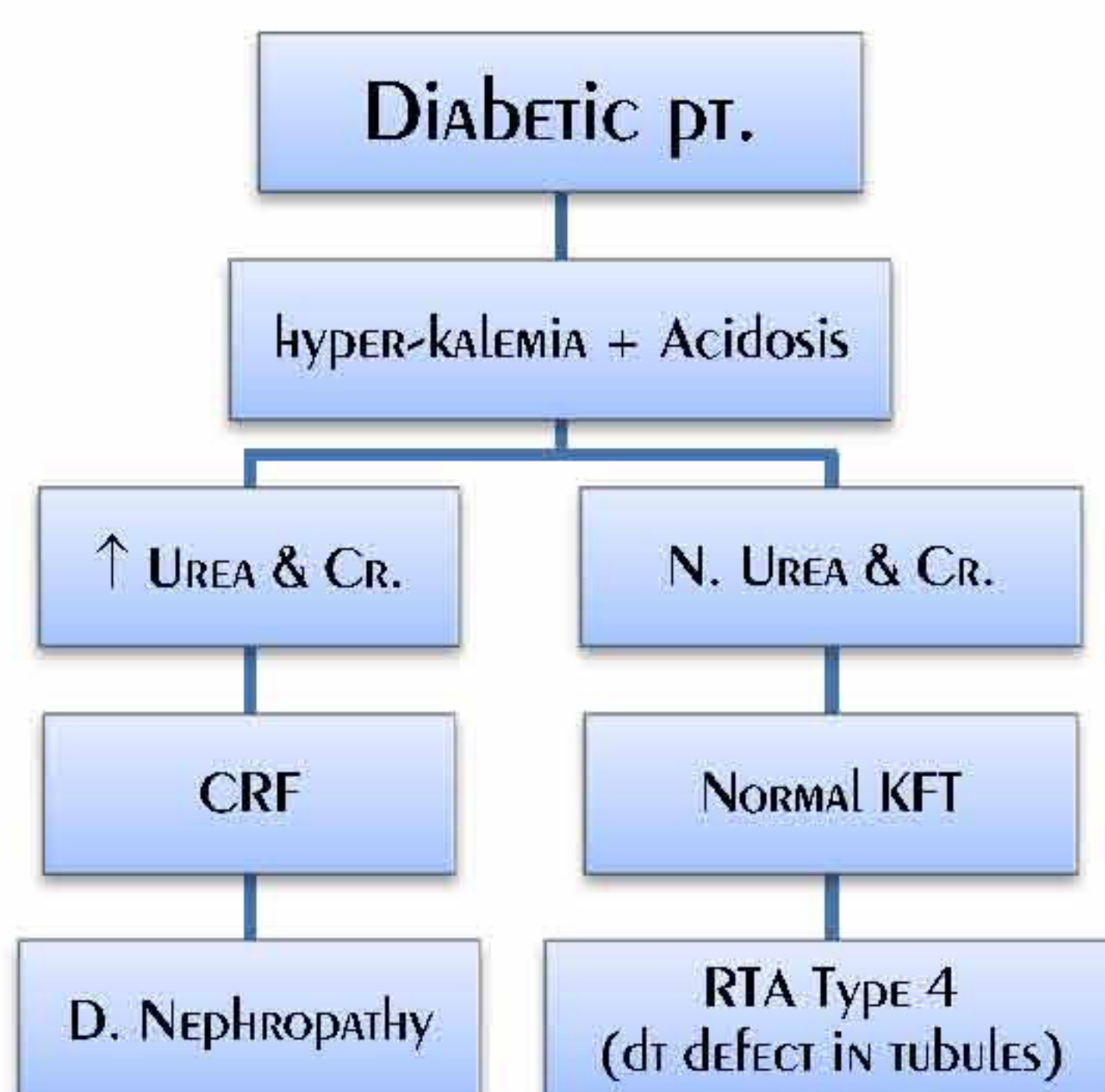
	TYPE 1 RTA	TYPE 2 RTA
PATHOLOGY	DCT defect $\rightarrow$ CAN'T SECRETE H^+ $\Rightarrow$ RETENTION OF H^+ 1) Acidosis $\rightarrow$ $\downarrow$ tubular reab. of Ca حموضة في الدم $\rightarrow$ Hypercalcuria $\rightarrow$ CA STONES. حصوات في الكلى 2) RICKETS ($\downarrow$ Ca in blood) $\Rightarrow$ Stunted gr. كساح 3) Tubules can't conc. Urine $\Rightarrow$ polyUREA & $\downarrow$ K.	PCT defect $\rightarrow$ $\downarrow$ HCO_3^- RE-ABSORP. <i>(Isolated / or part of Fanconi S)</i> 1) Acidosis. 2) $\downarrow$ K & HCO_3^- as it is excreted in urine as K Salts. 3) URINE is ALKALINE.
NH_4Cl TEST (H^+ load)	- Blood $\rightarrow$ acidosis URINE pH > 5.5 (bec. Kidney <u>CAN'T</u> excrete H^+)	$\downarrow$ URINE pH < 5.5 bec. Kidney <u>CAN</u> excrete H^+
Ttt.	$NaHCO_3$ + K supplement	Na HCO_3 + K supplement.

OTHERS:

- RENAL GLUCOSURIA** $\Rightarrow$ (AR) glucose in urine & normal bl. glucose. (PCT defect)
- CYSTINURIA** $\Rightarrow$ $\downarrow$ re-absorption of filtered cystine $\rightarrow$ $\uparrow$ Cystine In urine $\rightarrow$ cystine stone.
- ALPORT'S S (HEREDITARY NEPHRITIS) = HEMATURIA + DEAFNESS**
 - $\sigma > \phi$.
 - GN $\rightarrow$ haematuria & proteinuria $\rightarrow$ ESRD.
 - Nerve deafness.

RELATION BET. KIDNEY & DEAFNESS:

- Alport's S.
- Amino-Glycosides.



URINE ANALYSIS	
GN	INTERSTITIAL NEPHRITIS
1) RBCs > WBCs. 2) RBCs CASTS. 3) ALBUMINURIA > 1 gm	1) WBCs > RBCs. 2) WBCs CASTS. 3) < 1 gm DT TUBULAR DAMAGE.
-VE	GLUCOSURIA. PHASPHATURIA

DRUG INDUCED NEPHROPATHY

1) ARF:	
• PRE-RENAL:	- hypovolemia → Diuretics. (loop) ↓ RBF → ACE-I.
• RENAL	- ATN → Aminoglycosides. - ACUTE HS IN → Methicillin - Sulpha - NSAID.
• POST-RENAL:	- Clots of blood → Anticoagulants. - Crystalluria → Sulfa. - RETROPERITONEAL fibrosis → Methysergide. (↓HGE)
2) CRF	• Analgesic nephropathy (chronic interstitial nephritis).
3) Nephrotic S	- NSAID - ACE-I (Captopril in large dose for long duration) - Penicillamine - Gold.
4) ACUTE GN	• Rifampicin, Albumin infusion.
5) TUBULAR defects	- FANCONI S → Outdated Tetracycline. - DI → Lithium. "proph. For Manic depressive"
6) ACUTE TIN.	• ABS = Penicillin & Cephalosporins.
7) CHRONIC TIN.	• NSAID

Analgesic Nephropathy (Ingestion of 2-3 kg of the drug)

♀ E HEADACHE / ARTHRITIS / NEUROSIS → ANALGESIC ABUSE

1) NSAID & Aspirin → ↓PG → ↓ blood supply of the kidney.
Uncoupling of OP.

2) PARACETAMOL → ↓ GLUTATHIONE.

➤ Pathology: **CHRONIC TIN + papillary NECROSIS**

➤ CL. /P: CRF. ANEMIA. UTI.

➤ TREATMENT: AS CRF.

	renal Artery Stenosis	ADPKD	renal Carcinoma
DEF.	1-2 % of all cases of HTN YOUNG ♀ E SEVER HTN	Hereditary AD Poly-cystic Kidney Diseases. ccc. by renal cysts ± extra-renal (Liver & CVS)	Common malignant tumor. Affecting . Above age of 40 ys.
Path.	- fibro-muscular dysplasia ⇒ young ♀ - Atherosclerotic ⇒ old age	enlarged / multiple <u>not communicating</u> cysts compressing renal parenchyma.	Adeno-carcinoma (large cell) arising from PCT spread by direct, lymphatic & blood.
➤ CL/P			
	YOUNG ♀ E SEVER HTN 1) Malignant HTN. 2) Abdominal Bruit. 3) Fundus ⇒ hyper-sensitive retinopathy. 4) hypo-kalemic HTN. (dt ischemia → ↑renin → ↑Ang. II → 2nd hyper-aldosteronism → ↑K secretion → hypo-kalemia)	40 ys. e +ve FH 1) Dull aching loin pain. 2) HAEMATURIA dt hge in cyst or infection. 3) HTN dt ⊕ of RAS. 4) CRF. Normal Hb dt ↑EP secretion. 5) Sub-ARACHNOID hge. dt rupture of Berry Aneurysm.	1- early → painless Haematuria. عكس 2- Late → loin pain. } -Wilm's Tumor 3- Para-malignant \$:- • ↑ RENIN ⇒ HTN. • ↑ ALP. • ↑ PTH ⇒ ↑ Ca • ↑ EP ⇒ Polycythaemia.
➤ INVEST.			
	1) Duplex SCAN. ... if +ve 2) RENAL Angiography. 3) MRA ⇒ to avoid contrast nephropathy. 4) Rapid SEQUENCE IVP = soft t. shadow of kidney. 5) SONAR ⇒ small ischemic kidney. 6) RENAL SCAN ⇒ ↓ perfusion after ACE-I	1) IVP: spider leg app. (calyces are elongated & attenuated) 2) SONAR - CT → ENLARGED NODULAR KIDNEY ± POLYCYSTIC LIVER. Why to DIAGNOSE ADPKD ?!! <pre> graph TD Root[Why to DIAGNOSE ADPKD ?!!] --> Patient[PATIENT] Root --> Relatives[RELATIVES] Patient --> Comp[↓ Complications & PRESERVE KFs] Patient --> Drugs[RECENT DRUGS ↓ Cyst prog.] Relatives --> Age[> 20 ys.] Relatives --> Cases[+VE CASES] Age --> Screen[SCREENING by SONAR & CT SCAN] Cases --> Avoid[AS pt. + Avoid NEPHROTOXIC DRUGS] </pre> DD: hydronephrosis. – Pyonephrosis – Tumor - Solitary cyst.	1) IVP. 2) SONAR - CT SCAN → RENAL identification. 3) RENAL Angiography 4) Biopsy. So ↑ RBCs in ALL: • URINE → HEMATURIA. • BLOOD → polycythemia.
Ttt.	1) Anti-HTN. (BBs / CCB / Diuretics + NOT ACE-I) 2) Angioplasty ⇒ RA DILATED BY BALLOON CATHETER. ➤ ACE-I & ARBS ARE # IN BI-LATERAL RAS.	1) HTN → ACE-I. 2) Infections → QUINOLONES PENETRATE CYSTS. 3) Recently → DRUGS ↓ Cyst progression.	Nephrectomy.

renal stones

predisposing factors $\Rightarrow$ causes ~~to be investigated~~ to avoid recurrence.

- 1) **Metabolic causes:** (c below)
- 2) **Obst. & Infection** $\Rightarrow$ (EPITH + PUS DEBRIS) $\rightarrow$ NIDUS $\rightarrow$ DEPOSITION OF CRYSTALS $\rightarrow$ STONES
- 3) **Diet** $\Rightarrow$ ~~Excess~~ MILK & ITS PRODUCTS $\Rightarrow$ CA STONES
- 4) **Climate** $\Rightarrow$ HOT WEATHER + $\uparrow\uparrow$ SWEATING + NO DRINKING OF WATER
- 5) **1st RENAL DISEASE** $\Rightarrow$ POLYCYSTIC KIDNEY - MEDULLARY SPONGE KIDNEYS - RTA

	renal stones			
	Ca Oxalate	Ca Phosphate	Uric acid	Cystine
<u>Causes</u>	<u>HYPER-CALCAURIA</u> 1) Idiopathic 2) RTA 3) $\uparrow$ Na in diet or loop diuretic.	<u>HYPER-CALCAEMIA</u> • Sarcoidosis. • $\uparrow$ PTH • Vit D toxicity.	<u>HYPER-URICEMIA & HYPER-URICOSURIA</u> <u>HYPER-OxALURIA</u> <u>= MEASURE 24 hr.</u> Type 1 = AR (De $\uparrow$ production of Oxidate by the liver) Type 2 = Mal abs. S (mal-absorption $\rightarrow$ $\uparrow$ FFA $\rightarrow$ binds to Ca in intestine $\rightarrow$ $\uparrow$ free Oxidate Absorption)	Cystinuria.
<u>Pathology</u>	• Brownish. • mamillated.	• Whitish. • Laminated., in Alk. urine.	• Whitish. • Smooth.	
<u>Specific TTT.</u>	<u>Ca STONES IN GENERAL:</u> • 1 st hyper-calcauria $\Rightarrow$ Thiazides to $\downarrow$ Ca in URINE. • 1 st PTH $\Rightarrow$ SURGERY. • RTA $\Rightarrow$ Na CITRATE $\rightarrow$ Alk. URINE. <u>Ca Oxalate</u> $\Rightarrow$... + Cholestyramine + Mg Citrate.		1) Na CITRATE (Alkaline URINE) 2) Allopurinol "only stone that disappears completely"	1) Na CITRATE (Alkaline URINE) 2) Penicillamine .

NB: All stones maybe (single / multiple) & radio-opaque except pure uric acid stone is radio-lucent.
 Ca deposition in renal parenchyma $\Rightarrow$ nephro-calcinosis (hyper-PTH / Renal TB / Vit D toxicity / RTA)

	Cl./P of renal stones
--	------------------------------

O	M	U	M	I
<u>Obstruction</u>	<u>migration</u>	<u>ulceration</u>	<u>metaplasia</u>	<u>Infection</u>
↓	↓	↓	↓	↓
hydro-nephrosis loin pain	colic	haematuria *transient	malignancy	pyelonephritis *Recurrent-UTI

- 1) **Asymptomatic.**
- 2) **RENAL colic:** at loin radiates to groin.
- 3) **ARF (POST RENAL) OR CRF.**

INVESTIGATIONS

IMAGING

- 1) **X-RAY** $\Rightarrow$ All are radio-opaque except Pure Uric acid.
- 2) **IVP** $\Rightarrow$ back pressure, stone & stricture.
- 3) **SONAR** $\Rightarrow$ back pressure, stone, kidney.
- 4) **RENAL SCAN** $\Rightarrow$ reveals obstruction.

Lab invest.

- 1) URINE** *RBCs* → (painful haematuria)
 pus cell → 2° infection.
 Crystals → oxalate, P, urate.
- 2) 24h URINARY Ca** – urate · oxalate. + s. Ca & Uric A.
- 3) STONE Analysis.**

TREATMENT (↓ size of stones & prevent ftt)

MEDICAL	
---------	--

RENAL colic

- a) **BED REST – WARM THE LOIN.**
- b) **Anti-spasmodic** $\Rightarrow$ **ATROPINE.**
- c) **Analgesics** $\Rightarrow$ **PETHIDINE IM**
- d) **NSAID** $\Rightarrow$ **NEPHROTOXIC**

Infusion method by IV drip of
(Saline + atropine + papaverine)

STONES TTT. IN STABLE CASES

- 1) Fluids + Anti-spasmodic + Diuretics
- 2) Specific TTT. (من الجدول)

DIET FOR RENAL STONES:

- 1) ↑↑ fluids.
- 2) Ca stones ⇒ ↓ Milk & cheese.
- 3) Oxalate stones ⇒ ↓ spinach / rhubarb.
- 4) Uric stones ⇒ ↓ liver, kidney, sardines.

Surgical

INDICATIONS:

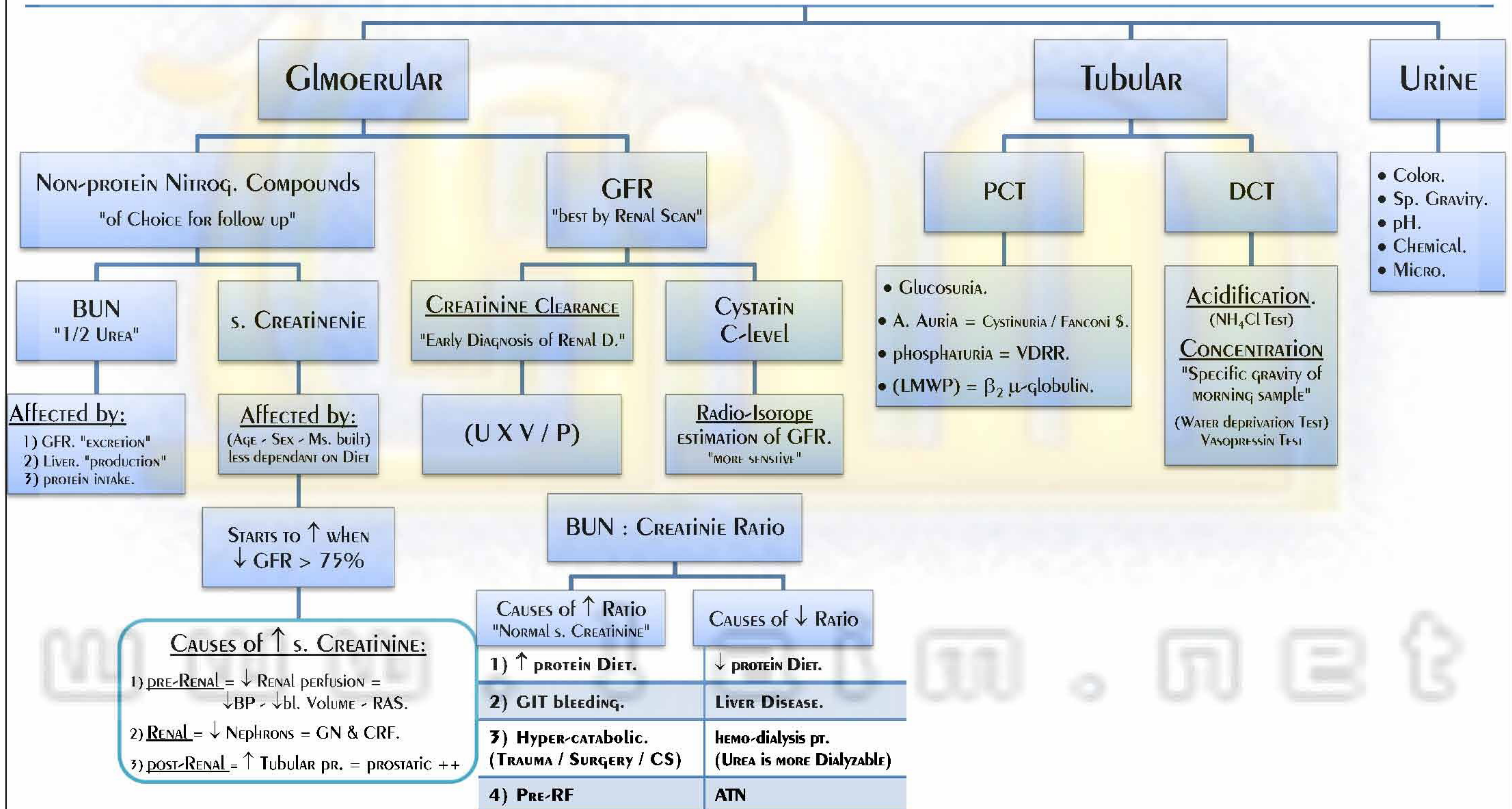
- 1) BIG / MULTIPLE STONES
- 2) PELVIC STONE / STAG-HORN.
- 3) HYDRONEPHROSIS

INTERFERENCE:

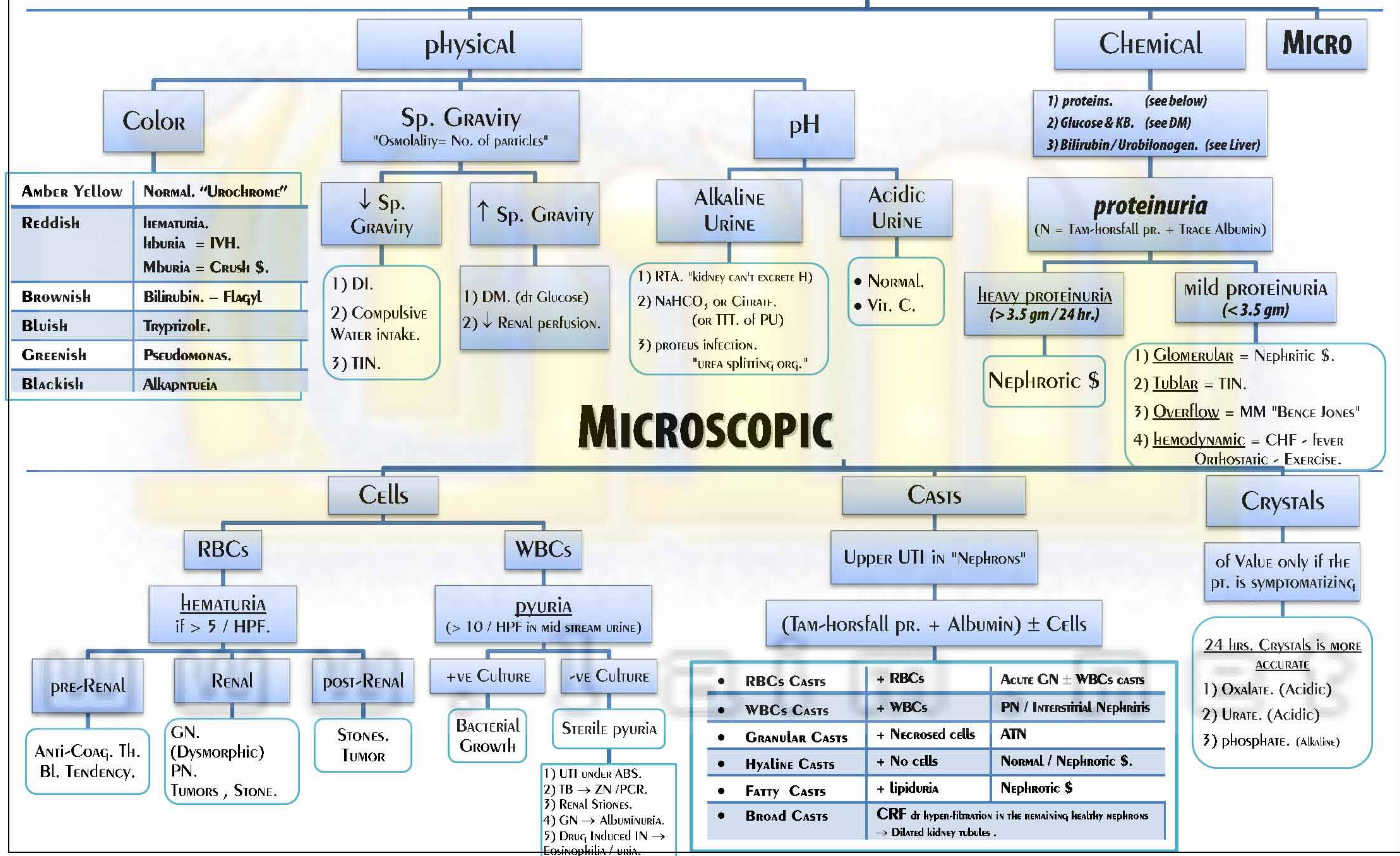
- 1) SURGERY.
- 2) ENDOSCOPY.
- 3) ESWL. (تفتيت الحصوة)

- 1) SURGERY.
- 2) Endoscopy.
- 3) ESWL. (تفتيت الحصوة)

KIDNEY FUNCTION TESTS (KFTs)



URINE ANALYSIS



Important Notes in Nephrology

p. 11	CONTRAST Nephropathy: (CT - Angiography – IVP)
	DM. Old Age RENAL D. ACE-I Multiple Myeloma Dehydration PRECAUTIONS: 1) <u>Good hydration.</u> 2) <u>Stop ACE-I</u> before it by 48 hrs. 3) <u>48 hrs b4 & After</u> ⇒ Aminophylline + NAC.
p. 20	Causes of CRF:
	1) Diabetic Nephropathy. (long duration) 2) Systemic HTN. (long duration) 3) Chronic GN. (history of puffiness + post-streptococcal infection) 4) Obstructive Uropathy. (Stones, B) 5) Analgesic Nephropathy. (Abuse of paracetamol + Aspirin) 6) Lupus Nephritis. (Young F + Arthropathy + Skin Rash) 7) Cong. Poly-cystic kidney. (+ve FH) 8) Gout. (history of Arthropathy + Allopurinol) 9) UN-determined etiology.
p. 22	CRF + ↓ BP
	1) Salt losing Nephropathy. Eg. TIN. 2) Hypo-reninism eg. (D. Nephropathy, Obst. Uropathy & IN)
	CRF + mild Anemia dt ↑ EP
	1) Polycystic Kidney. 2) Hyper-Nephroma & CRF. 3) Hydro-Nephrosis.
p. 41	MINIMAL LESION GN
	No CRF but ARF occurs due to excess Diuretics → hypovolemia → pre-RF

p. 46	Nephrotic \$ problems:
	1) Biharziasis → Nephrotic \$ → FSGS / MPGN. 2) Generalized LN++ → Nephrotic \$? - AIDS → FSGS. - lymphoma → minimal lesion 3) Fever → Nephrotic \$? - FMF → Amyloidosis kidney. - Malaria → Membranous GN. 4) Arthropathy → Nephrotic \$? - SLE → lupus Nephritis. - RA → Amyloidosis kidney. → Drugs → NSAID, penicillamine & Gold. 5) DM (Long-Standing) + Nephrotic \$ → Diabetic Nephropathy.
p. 63	Renal Calcification = Nephro-calcinosis
	- hyper-calcemia dt.....(hyper-PTH / Sarcoidosis / Vit. D Toxicity) - RTA - Renal TB. - Renal infarction. - RCC. (Renal Cell Carcinoma)
p. 72	Renal Vascular Disorders
• HTN	→ Nephrocalcinosis → CRF
• Malignant HTN	→ ARF.
• Scleroderma	Narrowing of Inter-lobular A. → Malignant Crisis → ARF.
• PAN	Narrowing of Arcuate A. → HTN
• TTP / HUS	Thrombocytopenia – hemolytic An. – RF
• Athero-Embolic Renal D.	- Catheter for Angioplasty → ulceration of Atheromatous plaque. - Anti-Coagulant + Thrombolytic Th. → showers of Cholesterol emboli → ARF + Systemic Emboli.
• <u>RV Thrombosis:</u> (CONGESTION Of Kidney → stretch of capsule → loin pain + RF)	- Nephrotic \$. (Membranous GN) - RCC. - Thrombophilia.

p. 75	Wilm's Tumor - Nephroblastoma
	➤ Cl./P: young child. (1st 3 ys.) • Early → Loin pain. • Late → Hematuria. ➤ INVEST.: SONAR – CT – Angiography. ➤ ITT.: Nephrectomy + Radio-th. "good response" "Early Diagnosis" عكس ال Renal carcinoma.
p. 76	LIVER D. → RF
	1) Hepato-Renal S. "in severe LCF → ARF" 2) B Nephropathy → FSGN & MPGN. 3) Viral hepatitis. 4) Rupture Varices Early → pre-RF. Late → ATN. 5) Obst. Jaundice → D. Bilirubin → Bilirubin Nephropathy.
p. 8	Albuminuria
	1) Normal Albumin in Urine < 30 mg/d. 2) μ - Albuminuria 30 - 300 mg/d. (early detection of D. Nephropathy) 3) Overt Albuminuria > 300 mg/d.
p. 8	Methods to measure proteinuria
	1) 24 hr. Urinary excretion. 2) Albumin / Cr. Ratio. 3) Dip-stick → CAN'T detect Bence Jones pr. of (Multiple Myeloma)

NORMAL VALUES:

• pH Urine	5-7
• Sp. Gravity	1003 – 1025
• Urinary proteins	150 – 200 mg / 24 hr.
• Pus cells	> 10 HPF.
• RBCs	> 5 HPF.
1) Bl. Urea	20 – 40 mg/dL
2) BUN	10-20 mg/dL
3) s. Creatinine	0.7 – 1.4
4) BUN : Creatinine	10 : 1